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Issue Date: 07 March 2006

Case No.: 2005-BLA-5182
2005-BLA-5183

In the Matter of:

Evelyn M. Childress, Widow
Clyde L. Childress, Deceased,
Claimant

v.

Tennessee Consolidated Coal,
Employer

And

Director, Office of Workers' Compensation Programs,
Respondent

DECISION AND ORDER DENYING BENEFITS ON LIVING MINER'S AND SURVIVOR'S CLAIMS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. Section 901 et seq. (the Act). In accordance with the Act and the regulations issued thereunder, the case was referred by the Director, Office of Workers' Compensation Programs for a formal hearing.

Benefits under the Act are awardable to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners who were totally disabled at the time of their deaths (for claims filed prior to January 1, 1982), or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as "black lung."

A formal hearing was held before the undersigned on October 19, 2005, in Knoxville, Tennessee, at which all parties were afforded full opportunity in accordance with the Rules of

Practice and Procedure (29 C.F.R. Part 18) to present evidence and argument as provided in the Act and the regulations issued thereunder, set forth in Title 20, Code of Federal Regulations, Parts 410, 718, 725, and 727.[1] At the hearing, I admitted Director's Exhibits 1 through 66, Administrative Law Judge Exhibits 1 and 2, Claimant's Exhibit 1, and Employer's Exhibits 1 through 15. The Claimant submitted her posthearing brief on January 11, 2006; the Employer submitted its posthearing brief on January 31, 2006; the Director did not submit a posthearing brief.

I have based my analysis on the entire record, including the exhibits and representations of the parties, and given consideration to the applicable statutory provisions, regulations, and case law, and made the following findings of fact and conclusions of law.[2]

JURISDICTION AND PROCEDURAL HISTORY

This case encompasses two separate claims. Mr. Clyde L. Childress filed a claim for black lung benefits on May 23, 2001, which was denied by the District Director on November 21, 2002 (DX 34). Mrs. Childress, on behalf of her husband, filed a request for modification, which was denied by the Director on September 30, 2003 (DX 37). Mrs. Childress again filed a request for modification on March 9, 2004, which was denied by the Director on August 23, 2004 (DX 38).

Mr. Childress died on August 17, 2003, and his widow, Evelyn M. Childress, filed a claim for survivor's benefits on October 6, 2003 (DX 41). On August 23, 2004, the Director issued a Proposed Decision and Order denying benefits (DX 61); by letter dated September 16, 2004, the Claimant appealed this determination and requested a hearing before an administrative law judge (DX 62).

Both claims were referred to the Office of Administrative Law Judges, and a hearing was held on October 19, 2005.

ISSUES PRESENTED

The issues contested by the Employer and the Director are:

b. Whether Mr. Childress had pneumoconiosis.
c. If so, whether Mr. Childress's pneumoconiosis arose out of his coal mine employment.

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1 The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vol. 65, No. 245 Wednesday, December 20, 2000. The revised Part 718 regulations became effective on January 19, 2001. Since the current claims were filed after January 19, 2001, the new regulations are applicable.

2 Citations to the record of this proceeding will be abbreviated as follows: "Tr." refers to the Hearing Transcript of the October 19, 2005 hearing; "ALJX" refers to the Administrative Law Judge's Exhibits; "DX" refers to the Director's Exhibits; "CX" refers to Claimant's Exhibit; and "EX" refers to Employer's Exhibits.

d. Whether Mr. Childress was totally disabled.
e. If so, whether Mr. Childress's total disability was due to pneumoconiosis.
f. Whether Mr. Childress's death was due to pneumoconiosis.

(DX 64; Tr. 22-23).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted and arguments made.

Background

The miner, Clyde L. Childress, was born on March 20, 1947, and died on August 17, 2003 (DX 41). He married his wife, Evelyn M. Childress, on DX August 13, 1966 (DX 2). At the time of his death, the miner was married and living with his wife, who has not remarried since the miner's death (Tr. 25). Mr. and Mrs. Childress do not have any children who are under the age of 18, or otherwise dependent (DX 2). I find that Mrs. Childress is the eligible survivor of the deceased miner, and that she has no dependents for purposes of augmentation of benefits.

Length of Coal Mine Employment

On his application for benefits, Mr. Childress indicated that he worked as a coal miner for 25 years, ending in 1993, when he was laid off (DX 2). Mrs. Childress, the Claimant, testified that her husband worked more than 23 years as a coal miner, from 1971 to 1993 (Tr. 25). The Director found that Mr. Childress had 19 years and 10 months of coal mine employment; the Employer agrees with this determination (Tr. 22-23). The Employer also agrees that it is correctly designated as the responsible operator. I find that Mr. Childress had at least 19 years and 10 months of coal mine employment, and that the Employer is properly designated as the responsible operator.

Mr. Childress's Deposition Testimony

Mr. Childress testified by deposition on December 28, 2001 (DX 32). Mr. Childress testified that he had been working for U.S. Pipe as a core remover since 1994. When he started work there, he took a physical. He was called back to take a tuberculosis test, and more x-rays. According to his testimony, when he went back three days later, everything had cleared up. However, he later found out from Dr. Ginsberg, his treating physician, that he did have tuberculosis. According to the Claimant, Dr. Ginsberg had performed a biopsy and x-rays, and told him he had black lung.

Mr. Childress testified that his breathing had gotten worse over the years. He had trouble when it was hot, or if the weather was cold. He also coughed a lot at night.

Evidentiary Issues

Both of the claims in this case fall under the evidentiary restrictions of the new regulations. Accordingly, I have considered the evidence separately designated by the parties in making a determination on Mr. Childress's living miner's claim, as well as Mrs. Childress's survivor's claim.

Mrs. Childress's Survivor's Claim

Applicable Standard

The Regulations at 20 C.F.R. § 718 apply to survivors' claims which are filed on or after April 1, 1980. 20 C.F.R. § 718.1. Because Mrs. Childress filed her survivor's claim after January 1, 1982, 20 C.F.R. § 718.205(c) applies to this claim.

The regulations provide that a survivor is entitled to benefits only where the miner died due to pneumoconiosis. 20 C.F.R. § 718.205(a). The Claimant must establish that: (1) the decedent was a coal miner; (2) the decedent suffered from pneumoconiosis at the time of his death; (3) the decedent's pneumoconiosis arose out of his coal mine employment; and (4) the decedent's death was caused by pneumoconiosis or pneumoconiosis was a substantially contributing cause or factor leading to his death. All elements of entitlement must be established by a preponderance of the evidence. *Strike v. Director, OWCP,* 817 F.2d 395, 399 (7th Cir. 1987). The survivor of a miner who was totally disabled due to pneumoconiosis at the time of death, but died due to an unrelated cause, is not entitled to benefits. 20 C.F.R. § 718.205(c). If the principal cause of death is a medical condition unrelated to pneumoconiosis, the survivor is not entitled to benefits unless the evidence establishes that pneumoconiosis was a substantially contributing cause of the death. 20 C.F.R. § 718.205(c)(4).

X-ray Evidence

Exhibit No.	***Date of X-Ray***	***Date of Reading***	***Physician/ Qualifications3***	***Interpretation***
DX 12	7-20-01	7-20-01	Hudson	2/3, q, t
DX 12	7-20-01	9-11-01	Sargent/B, BCR	Read for quality purposes only
DX 14	7-20-01	11-19-01	Wheeler/B, BCR	0/1, q, t
DX 19	11-15-02	4-11-03	Scatarige/B, BCR	Negative for pneumoconiosis
DX 48	11-15-02	12-13-02	Ahmed/B, BCR	3/3
CX 2	11-15-02	12-20-02	Pathak/B, BCR	2/3, r, u, Category B opacities
EX 1	11-15-02	4-11-03	Wheeler/B, BCR	0/1, t, q
DX 48	8-7-03	8-8-03	Powell	Chronic interstitial lung disease;

3 A "B-reader" is a physician, but not necessarily a radiologist, who has successfully completed an examination in interpreting x-ray studies conducted by, or on behalf of, the Appalachian Laboratory for Occupational Safety and Health (ALOSH). A designation of "BCR" means that the physician is "certified" in radiology or diagnostic roentgenology by the American Board of Radiology or the American Osteopathic Association.

				interval development in diffuse opacity throughout left lung field
DX 48	8-8-03	8-9-03	Parker	Bilateral diffuse infiltrates
DX 48	8-9-03	8-9-03	Parker	Bilateral diffuse infiltrates
DX 48	8-13-03	8-13-03	Ho	Bilateral airspace disease

Autopsy/Biopsy Reports

Dr. Lori D. Emerson

Dr. Emerson performed an autopsy that was limited to a biopsy of the lungs (DX 46). She removed a wedge of lung tissue, which she described as grossly black and firm, with evidence of pleural thickening.

Dr. Emerson's final anatomic diagnosis was complicated coal workers' pneumoconiosis. She noted that the lung biopsy showed extensive scarring, with heavy black pigment deposition. The areas of scarring showed central necrosis; there was emphysema adjacent to the large scars.

Dr. Gary R. Lanham

Dr. Lanham reviewed two slides from the right lung wedge biopsy performed after Mr. Childress's death on August 17, 2003 (CX 1). He reported that there was extensive formation of silicotic nodules, and deposition of dust compatible with coal dust. He noted that the slides showed replacement of much of the pulmonary parenchyma by large fibrotic nodules. In many areas, the features were characteristic of silicotic nodules, with formation of dense circular collagen nodules containing large amounts of birefringent material, compatible with quartz (crystalline silica). There were also multiple fragments of transparent brown or amber colored crystal like material, suggestive of coal dust. He stated that the carbon dust from coal itself is generally not the cause of pulmonary disease. Rather, the accompanying silica material is generally the problem, in the formation of fibrous nodules. He stated that "some authorities seen to lump the changes seen here under the general term of coal workers' pneumoconiosis, while others appear to split the findings into two separate categories."

Dr. Richard L. Naeye

Dr. Naeye reviewed medical records at the Employer's request, and also examined the autopsy slides (DX 47). He noted that Mr. Childress worked as an underground coal miner for 23 years, mostly as a roof bolter. He had a 25-50 pack year history of cigarette smoking, and quit many years before he died. Mr. Childress's chest x-rays in recent years had usually described diffuse, micronodular, macronodular lung disease with interstitial features, which was most often attributed to histoplasmosis or tuberculosis. In May 2003, an atypical mycobacterium was cultured from his lungs. His pulmonary function studies in later years consistently produced low FEV1, FVC, and MVV values.

According to Dr. Naeye, Mr. Childress had a twenty year history of hypertension. On August 8, 2003 he went to the emergency room with complaints of chest pain. En route to another hospital, he had two episodes of cardiac arrest; on admission it was found that he had a large, recent anterior lateral myocardial infarction. His cardiac and renal failure progressed, and he died on August 17, 2003. His death was attributed to the large myocardial infarction and underlying coronary artery disease. An autopsy of his lungs was performed, and the pathologist attributed the granulomatous lesions to complicated coal workers' pneumoconiosis.

On examination of the two autopsy slides, Dr. Naeye noted that most of the lung tissue was replaced with large lesions, mainly comprised of active granulation tissue, with degenerated areas in some of their centers. He noted a very small amount of black pigment admixed with granulation tissue at most sites. The birefringent crystals were few in number in most areas of the lesions. Almost all of the crystals were in a size range that classified them as non-toxic silicates. Dr. Naeye noted that very tiny birefringent crystals of toxic free silica were so rare that there was no possibility they could have had any role in the genesis of the large granulomatous lesions. According to Dr. Naeye, the rare presence of these tiny crystals was a clear indication that the lesions were non-silicotic in origin. He noted only a very small amount of black pigment in very tiny deposits in the few areas of lung tissue where the granulomatous lesions were absent. Very tiny birefringent crystals of toxic free silica were rarely associated with the pigment.

Dr. Naeye felt that there was not enough lung tissue available for him to evaluate the severity of any emphysema that could be in Mr. Childress's lungs. There were no bronchi or bronchioles available for microscopic review, so he could not determine the presence or absence of chronic bronchitis and bronchiolitis by microscopic review.

Dr. Naeye concluded that most of the lung tissue available for microscopic review was replaced by large granulomatous lesions of possible infectious origin. He felt that the histologic findings, and their widespread distribution did not support the autopsy prosector's diagnosis of complicated pneumoconiosis. According to Dr. Naeye, the lesions of complicated pneumoconiosis are usually localized, and most often in the upper lobe of one or both lungs. But the lesions in Mr. Childress's case were widely spread, and thus they were likely infectious in origin. In addition, there was very little black pigment in the lesions, and no very tiny birefringent crystals of toxic free silica, or blood vessel destruction with resultant large central areas of necrosis. He stated that this latter feature was characteristic for complicated pneumoconiosis, which presents a rather narrow outer zone of active granulation tissue, and large hyalinized and/or necrotic centers. But the massive lesions in Mr. Childress's lungs were mainly composed of granulation tissue, with only occasional small hyalinized or necrotic foci. According to Dr. Naeye, there is always plentiful black pigment in lesions of complicated pneumoconiosis, but there was very little such pigment in Mr. Childress's lung lesions. Finally, the lesions were not silicotic in origin, because there were only very rare tiny birefringent crystals of free silica in them.

Dr. Naeye felt that there were not minimum findings of simple pneumoconiosis in the lung tissues provided for his microscopic review. He stated that to make such a diagnosis, there must be black pigment present at characteristic subpleural and peribronchial loci, with accompanying evidence of tissue damage. But there were not such lesions in the lung tissues he

reviewed. Because there was no pneumoconiosis, it could not have caused any measurable abnormalities in lung function, or any disability, or contributed in any way to Mr. Childress's death.

Dr. P. Raphael Caffrey

Dr. Caffrey reviewed medical records at the Employer's request, and submitted a report dated April 28, 2004 (EX 2). He also examined two slides from Mr. Childress's autopsy.

On microscopic examination of the first tissue slide, Dr. Caffrey noted that all of the lung tissue showed areas of granulation with fibrinoid material within the alveoli in many areas. The largest piece showed large areas of scarring with dense collagen, with some anthracotic pigment, and some necrosis within the areas of scar tissue with pigment in at least one area. Some of the areas of scar were greater than 1 cm. in size. There was also a mild degree of centrilobular emphysema. He stated that under polarized light, there were a few birefringent particles, which he felt were consistent with silica. There were no macules, or coal dust with reticulin and focal emphysema, and no micronodules. He saw no true granulomas, or giant cells.

The second slide contained two very large pieces of lung tissue, both of which had large areas of irregular scarring with some anthracotic pigment. Dr. Caffrey noted that there were macrophages and some fibrinoid material and/or hyaline membrane material in many of the alveoli, as well as mucous and inflammatory cells in many of the bronchioles. Dr. Caffrey did not see any distinct granulomas consisting of epithelial cells, mononuclear cells, and giant cells. He noted a mild infiltrate of mononuclear cells at the periphery in a number of areas. But there were no true macules or micronodules. Dr. Caffrey found a mild degree of centrilobular emphysema. Under polarized light, he saw a few birefringent particles, which he felt were consistent with silica.

Dr. Caffrey's final diagnosis was multiple foci of granulation tissue and scar tissue, mild amount of anthracotic pigment, and mild centrilobular emphysema.

After reviewing Mr. Childress's records, Dr. Caffrey felt that it was quite clear that he had a cardiac death, and suffered a large myocardial infarction, due to coronary artery disease. He stated that there was no relationship between Mr. Childress's coal mining employment and his death due to myocardial infarction.

Dr. Caffrey felt that there was no question that Mr. Childress developed mycobacterium avium and/or mycobacterium simae, for which he was treated. According to Dr. Caffrey, these are some of the most common opportunistic mycobacteria, and infection usually comes from the environment, in contrast to mycobacterium tuberculosis, which is almost always transmitted from an infected individual. But he was not aware of any association or relationship between mycobacterium aviae or simae and pneumoconiosis. He stated that there were definitely no findings on the slides to suggest silicosis.

In Dr. Caffrey's opinion, the biopsy slides showed the presence of anthracotic pigment, which was compatible with Mr. Childress's history of 21 years working in the coal mines. These

slides showed granulation tissue and scar tissue, which could well be the result of Mr. Childress's previous drug treatment for the mycobacterium organisms. He felt that the amount of autopsy material available was limited, and he could not make an absolute diagnosis. According to Dr. Caffrey, the classical microscopic findings of complicated pneumoconiosis were not on the slides, and the typical finding of granulomas, which consists of epithelial cells, mononuclear cells, and giant cells, was also not present. In his opinion, the lack of those findings made it difficult to make a specific diagnosis. There were no macules or micronodules of pneumoconiosis on the slides, and the lack of these findings were against a finding of complicated pneumoconiosis. He felt that it was unfortunate that Mr. Childress did not have a complete autopsy at least involving both lungs.

Medical Reports/Treatment Records

Dr. Joel Ginsberg

Dr. Ginsberg's treatment notes, covering the period of December 2002 to March 2003, are in the record (DX 19). In his December 29, 2002 note, Dr. Ginsberg noted that Mr. Childress was a smoker, with longstanding exposure to a foundry environment, including coal dust. He indicated that bilateral interstitial infiltrates were considered compatible with pneumoconiosis. Mr. Childress was a remote smoker with mild dyspnea in the past. He had been admitted to the Erlanger Medical Center in February 2001 for toxic symptoms of right apical pulmonary abscess, which was confirmed as mycobacterium avium, and which responded to antibiotics.

According to Dr. Ginsberg, as part of an evaluation for impairment associated with pneumoconiosis, Dr. Snell had reviewed Mr. Childress's x-rays and suggested an evaluation for mycobacterial disease. Mr. Childress had indicated that he had profound weakness and progressive dyspnea over several months. Sputum specimens obtained the preceding week showed AFB organisms on all three specimens.

On examination of Mr. Childress, Dr. Ginsberg noted severely diminished breath sounds in all fields, and crepitant fine rales in the posterior lung zones. Mr. Childress described significant dyspnea on exertion. His chest x-ray showed a modest progression of nodular infiltrate in the lung fields, particularly the left axillary region and apex. Pneumoconiosis was noted on previous films.

Dr. Ginsberg recommended that Mr. Childress begin therapy directed at tuberculosis and/or mycobacterium avium complex. On December 31, 2002, Mr. Childress was seen at the Department of Health Tuberculosis Control Clinic. Dr. Adam Soufleris reported that he was probe negative for mycobacterium tuberculosis, but he suspected that he had mycobacterium avium, because he had had it before. According to Dr. Soufleris, Mr. Childress had significant lung disease and a positive skin test for tuberculosis. He felt that Mr. Childress would benefit from a course of prophylactic therapy.

Dr. Ginsberg saw Mr. Childress on January 31, 2003. At that time, Mr. Childress described progressive dyspnea on exertion, productive increasing cough, and myalgia. A CT scan performed on January 2, 2003 showed progression of disease in the lungs, with marked

atelectasis, cavitation in the left upper lobe, increasing fibrosis and atelectasis in the right lung field. He noted military disease in the lung bases, especially on the left. There was a large cavitary lesion at the level of carina, measuring 3.5 cm., and extending towards the apex, where it reached 5 cm. There was air bronchogram in the apical posterior segment, and increasing disease in the right upper lobe, with lesser degree of involvement in both lower and right middle lobes. There was no cavitation on the right, but increasing fibrosis and atelectasis. Multiple military nodules were seen in the left lower lobe. There was no pleural effusion, or identifiable mass lesions. Right paratracheal and hilar adenopathy, and calcified bilateral hilar and mediastinal nodes were identified. Active tuberculosis was considered.

On examination of Mr. Childress, Dr. Ginsberg noted fine rales throughout his lung fields bilaterally. Chest x-rays showed progression of disease, particularly on the left. He identified micronodular and macronodular lesions that spared no area of the lung.

Dr. Ginsberg stated that Mr. Childress presented with pneumoconiosis associated with dust inhalation, as well as mycobacterium simiae, and organism that behaves similarly to mycobacterium avium cellularae. He recommended drug therapy.

Dr. Ginsberg saw Mr. Childress on March 3, 2003, and noted that he had experienced minimal improvement. Mr. Childress described significant dyspnea on exertion. Dr. Ginsberg noted a modest improvement in infiltrates shown on x-ray.

Dr. Morrow Chamberlain II

Dr. Chamberlain saw Mr. Childress at Dr. Ginsberg's request (DX 19). He noted Mr. Childress's 4 to 6 month history of difficulty swallowing, dysphagia, and frequent choking episodes. Dr. Chamberlain noted that Mr. Childress was diagnosed with tuberculosis about a year earlier, but discontinued his medication after his discharge from the hospital. He had been on therapy for about three weeks.

After examination of Mr. Childress, Dr. Chamberlain concluded that he had left vocal cord paralysis, and tuberculosis.

Hospitalization Records

Mr. Childress was admitted to the Parkridge Medical Center on August 8, 2003 (DX 48). Dr. Joel Ginsberg, who treated him at the hospital, recorded admitting diagnoses of respiratory failure on mechanical ventilator, presumed pneumoconiosis with severe upper lobe scarring, and mycobacterium simeon pulmonary infection on antimycobacterial therapy. At the time of Mr. Childress's death, Dr. Ginsberg reported diagnoses of anterolateral myocardial infarction with severe ischemic cardiomyopathy, respiratory failure on mechanical ventilator, presumed pneumoconiosis, previous mycobacterium infection with cavitary lung lesions, and diffuse pulmonary infiltrates, a likely combination of lung injury and pulmonary edema.

Dr. Ginsberg noted that Mr. Childress had a history of chronic lung disease from mycobacterium simeon on antituberculous therapy, and presumed pneumoconiosis. He was

brought to the emergency room with severe chest pain and shortness of breath. On examination of Mr. Childress, Dr. Ginsberg noted that his lungs showed bilateral coarse rhonchi. His chest x-ray showed marked pleural thickening in the upper lung fields, with some cavitary lesions and diffuse pulmonary infiltrates.

Mr. Childress was administered antibiotics. His echocardiogram showed severe cardiomyopathy, with an ejection fraction of 20%. He underwent angioplasty, but continued to deteriorate. He died on August 17, 2003.

Dr. Daniel E. Hood Jr. saw Mr. Childress in consultation on August 13, 2003. He noted Mr. Childress's history of multilobar pneumonia, interstitial lung disease, hyperglycemia, normocytic normochromic anemia, hypertension with diastolic dysfunction, and ischemic cardiomyopathy. Dr. Hood noted diffuse bilateral rhonchi in Mr. Chidress's lungs. His impression was ischemic cardiomyopathy; he indicated the feasibility of cardiac surgery.

Dr. Daniel E. Constantinescu saw Mr. Childress in consultation on August 8, 2003. He noted that Mr. Childress was on medication for treatment of tuberculosis. Dr. Constantinescu noted rhonchi, secretions, and rattling sounds on examination of Mr. Childress's lungs. His impression was acute anteroseptal lateral MI with severely depressed LV function, probable multivessel disease, chronic lung disease with mycobacterium avium, and hemodynamic instability. He did not feel that Mr. Childress was a candidate for intervention, because his pulmonary condition was very severe, and he had poor LV function. According to Dr. Constantinescu, Mr. Childress's chronic pulmonary condition was a very serious setback in his situation.

Dr. James D. Snell

Dr. Snell prepared a report dated May 6, 2003, after reviewing Mr. Childress's CT scan (DX 20). According to Dr. Snell, it confirmed a pattern more consistent with granulomatous infection than pneumoconiosis. He also indicated that at his request, sputum cultures were obtained, which showed that Mr. Childress had a recurrent infection with inadequately treated mycobacterium simae. He stated that this was a very rare tuberculosis related organism, with no good definition of appropriate therapy. Although Mr. Childress was being treated with the best treatment program from information available in the medical literature, not enough time had passed to determine if he would respond.

Dr. Snell felt that it was very clear that Mr. Childress did not suffer from an occupational problem. Rather, he suffered from an infection that had flared up for years, and gotten out of control, producing major impairment and disability.

Death Certificate

Dr. Nathan Mull completed the death certificate on August 21, 2003 (DX 44). He indicated that the immediate cause of Mr. Childress's death was ventricular tachycardia progressing to asystole, due to large myocardial infarction and coronary artery disease. As other significant conditions contributing to death but not resulting in the underlying cause, he listed

pneumoconiosis, and atypical mycobacterial pulmonary infection. Dr. Mull indicated that he did not have the autopsy findings available.

Discussion

The Regulations at 20 C.F.R. § 718 apply to survivors' claims which are filed on or after April 1, 1980. 20 C.F.R. § 718.1. Because the Claimant filed her survivor's claim after January 1, 1982, 20 C.F.R. § 718.205(c) applies to this claim.

The regulations provide that a survivor is entitled to benefits only where the miner died due to pneumoconiosis. 20 C.F.R. § 718.205(a). The Claimant must establish that: (1) the decedent was a coal miner; (2) the decedent suffered from pneumoconiosis at the time of his death; (3) the decedent's pneumoconiosis arose out of his coal mine employment; and (4) the decedent's death was caused by pneumoconiosis or pneumoconiosis was a substantially contributing cause or factor leading to his death. All elements of entitlement must be established by a preponderance of the evidence. *Strike v. Director, OWCP,* 817 F.2d 395, 399 (7th Cir. 1987). The survivor of a miner who was totally disabled due to pneumoconiosis at the time of death, but died due to an unrelated cause, is not entitled to benefits. 20 C.F.R. § 718.205(c). If the principal cause of death is a medical condition unrelated to pneumoconiosis, the survivor is not entitled to benefits unless the evidence establishes that pneumoconiosis was a substantially contributing cause of the death. 20 C.F.R. § 718.205(c)(4).

The Board has held that death will be considered to be due to pneumoconiosis where the cause of death is significantly related to or significantly aggravated by pneumoconiosis. *Foreman v. Peabody Coal Co.,* 8 B.L.R. 1-371 (1985). The United States Court of Appeals for the Sixth Circuit, in which the instant case arises, has held that pneumoconiosis is a substantially contributing cause of death if it hastens, even briefly, the miner's death. *See, Brown v. Rock Creek Mining Corp.,* 996 F.2d 812 (6th Cir. 1993)(J. Batchelder dissenting). *See also, Shuff v. Cedar Coal Co.,* 967 F.2d 977 (4th Cir. 1992), *cert. denied,* 113 S.Ct. 969 (1993); *Peabody Coal Co. V. Director, OWCP,* 972 F.2d 178 (7th Cir. 1992); *Lukosevicz v. Director, OWCP,* 888 F.2d 1001 (3rd. Cir. 1989).

The Board has held that in a Part 718 survivor's claim, the Judge must make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. § 718.202(a) before considering whether the miner's death was due to the disease under § 718.205. *Trumbo v. Reading Anthracite Co.,* 17 B.L.R. 1-85 (1993).

Existence of Pneumoconiosis

Pneumoconiosis is defined, by regulation, as a "chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." 20 C.F.R. § 718.201. The regulations at 20 C.F.R. § 718.203(b) provide that, if it is determined that the miner suffered from pneumoconiosis and engaged in coal mine employment for ten years or more, there is a rebuttable presumption that the pneumoconiosis arose out of such employment. If, however, it is established that the miner suffered from pneumoconiosis but worked less than ten years in the coal mines, then the claimant must

establish causation by competent evidence. *Stark v. Director, OWCP,*9 B.L.R. 1-36 (1986); *Hucker v. Consolidation Coal Co.,*9 B.L.R. 1-137 (1986). The Board has held that the burden of proof is met under § 718.203(c) where "competent evidence establish(es) that his pneumoconiosis is significantly related to or substantially aggravated by the dust exposure of his coal mine employment." *Shoup v. Director, OWCP,* 11 B.L.R. 1-1101-112 (1987). Specifically, the record must contain *medical* evidence to demonstrate causation. *Baumgartner v. Director, OWCP,* 9 B.L.R. 1-65, 1-66 (1986)(administrative law judge cannot infer causation based solely upon claimant's employment history); *Tucker v. Director, OWCP,* 10 B.L.R. 1-35, 1-39 (1987)(it was error for the administrative law judge to rely solely upon lay testimony to find causation established).

The existence of pneumoconiosis may be established by any one or more of the following methods: (1) chest x-rays; (2) autopsy or biopsy; (3) by operation of presumption; or (4) by a physician exercising sound medical judgment based on objective medical evidence. 20 C.F.R. § 718.202(a).

X-Ray Evidence

In this case, Dr. Hudson interpreted Mr. Childress's July 20, 2001 x-ray as positive for pneumoconiosis. Dr. Hudson is neither a B reader nor a board certified radiologist. Dr. Wheeler, who is both, interpreted this x-ray as negative. Relying on Dr. Wheeler's superior qualifications, I find that this x-ray is negative for pneumoconiosis.

There are four interpretations, all by dually qualified readers, of an x-ray performed on November 15, 2002. Dr. Ahmed and Dr. Pathak both interpreted this x-ray as positive for pneumoconiosis. However, Dr. Scatarige and Dr. Wheeler interpreted it as negative. Given the equal balance of positive and negative interpretations by physicians who are all dually qualified, I find that these interpretations are in equipoise, and thus this x-ray does not establish the existence of pneumoconiosis.

The remaining x-ray interpretations are part of the hospitalization records from Mr. Childress's final admission. None of these interpretations contain findings of pneumoconiosis. Dr. Parker noted bilateral diffuse infiltrates, and Dr. Ho reported bilateral airspace disease. Dr. Powell reported findings of chronic interstitial lung disease, and an interval development in a diffuse opacity throughout the left lung field. But none of them indicated that these findings were consistent with pneumoconiosis. I find that these x-ray reports are not sufficient to establish the existence of pneumoconiosis.

There are no remaining x-ray interpretations in the record of the survivor's claim. I find that the Claimant has not established the existence of pneumoconiosis by a preponderance of the x-ray evidence.

Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found applicable. In the instant case, the presumption of § 718.305 does not apply to claims filed after January 1, 1982. Section 718.306 does not apply to claims where the miner died after March 1, 1978. Section 718.304 allows a presumption of

complicated pneumoconiosis where, *inter alia*, an x-ray "yields one or more large opacities (greater than 1 centimeter in diameter) and would be classified in Category A, B, or C" if such miner is suffering or suffered from a chronic dust disease of the lung. 20 C.F.R. § 718.304(a). However, if the employer can affirmatively show the opacity is something other than pneumoconiosis, the x-ray loses force, and the claimant loses the benefit of the presumption. *See Eastern Associated Coal Corp. v. Director, OWCP [Scarbro]*, 220 F.3d 250, 256 (4th Cir. 2000).

Both Dr. Ahmed and Dr. Pathak interpreted Mr. Childress's November 15, 2002 x-ray as showing Category B opacities. Neither Dr. Scatarige nor Dr. Wheeler read this x-ray as showing pneumoconiosis, but both noted extensive fibrosis and pleural thickening, findings that do not affirmatively establish that the process designated as Category B opacities by Dr. Ahmed and Dr. Pathak was not in fact present. However, the remaining medical records, including the results of extensive laboratory tests, establish overwhelmingly that Mr. Childress suffered from a rare form of tuberculosis. I find that this evidence affirmatively establishes that the processes in Mr. Childress's lungs, as seen on x-ray, are the result of his tuberculosis, but not pneumoconiosis. Accordingly, the Claimant has not established the existence of pneumoconiosis by virtue of § 718.202(a)(3).

Autopsy/Biopsy Evidence

Dr. Emerson removed a wedge of lung tissue after Mr. Childress's death, which she described as grossly black and firm, with evidence of pleural thickening. She concluded that Mr. Childress had complicated pneumoconiosis, pointing to extensive scarring with central necrosis, and heavy black pigment deposition.

Dr. Caffrey viewed the slides from Mr. Childress's lung tissue, and concluded that there were definitely no findings to suggest silicosis. On his microscopic examination of both slides, he found a few birefringent particles, consistent with silica, but no true macules or micronodules, or coal dust with reticulin and focal emphysema. His final diagnosis was multiple foci of granulation and scar tissue, a mild amount of anthracotic pigment, and mild centrilobular emphysema.

According to Dr. Caffrey, the amount of autopsy material was limited, and thus he was not able to make an absolute diagnosis. But he found no macules or micronodules of pneumoconiosis on the tissue slides, which argued against a finding of complicated pneumoconiosis. He acknowledged the presence of anthracotic pigment, as well as granulation and scar tissue, which he felt could be the result of Mr. Childress's drug treatment for the mycobacterium organisms. But the classical microscopic findings of complicated pneumoconiosis were not on the slides.

Dr. Naeye also examined the autopsy slides, and concluded that there were no findings of simple pneumoconiosis. He noted that on both slides, most of the lung tissue had been replaced with large lesions mainly comprised of active granulation tissue of possible infectious origin, with degenerative areas at some of the centers. He found only a very small amount of black pigment mixed with the granulation tissue, and few birefringent crystals. He noted that almost

all of the crystals were of a size that classified them as non-toxic silicates. Their rare presence was a clear indication that the lesions were non-silicotic in origin.

Dr. Naeye did not agree with Dr. Emerson's diagnosis of complicated pneumoconiosis, noting that lesions of complicated pneumoconiosis are usually localized, and most often in the upper lobes. But the lesions in Mr. Childress's case were widely spread, and thus likely infectious in origin. Moreover, there was very little black pigment in the lesions, and no very tiny birefringent crystals of toxic free silica, or blood vessel destruction with resultant large central areas of necrosis, which was characteristic of complicated pneumoconiosis. Rather, the massive lesions were mainly composed of granulation tissue, with only occasional small hyalinized or necrotic foci. But in lesions of complicated pneumoconiosis, there was always black pigment, whereas there was very little pigment in these lesions. Finally, the lesions were not silicotic in origin, because there were only very rare tiny birefringent crystals of free silica in them.

According to Dr. Naeye, in order to diagnose simple pneumoconiosis, there must be black pigment at characteristic subpleural and peribronchial loci, with accompanying evidence of tissue damage. But there were not such lesions in the tissues he reviewed.

In weighing these autopsy/biopsy reports, I place most reliance on the opinions of Dr. Caffrey and Dr. Naeye, which I find to be thorough, well-reasoned, and supported by the objective evidence. Both doctors examined the tissue slides microscopically, and described their findings in detail, explaining why they were not consistent with pneumoconiosis, simple or complicated. In contrast, Dr. Emerson did not view the tissue slides microscopically. Her report is brief and conclusory, and she did not explain why or how her findings of extensive scarring with central necrosis, pigment deposition, and emphysema supported a finding of complicated pneumoconiosis.

Relying on the reports by Dr. Caffrey and Dr. Naeye, I find that the Claimant has not established the existence of pneumoconiosis by a preponderance of the autopsy/biopsy evidence.

A claimant can also establish that the miner suffered from pneumoconiosis by well-reasoned, well-documented medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. *Fields v. Island Creek Coal Co.,* 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's history. *See, Hoffman v. B&G Construction Co.,* 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.,* 7 B.L.R. 1-295 (1984). A report which is better supported by the objective medical evidence of record may be accorded greater probative value. *Minnich v. Pagnotti Enterprises, Inc.,* 9 B.L.R. 1-89, 1-90 n.1 (1986); *Wetzel v. Director, OWCP,*8 B.L.R. 1-139 (1985).

A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. *Fields, supra.* Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder of fact to decide. *Clark v. Karst-Robbins Coal Co.,* 12 B.L.R. 1-149 (1989)(en banc). Statutory pneumoconiosis is established by well-reasoned medical reports which support a

finding that the miner's pulmonary or respiratory condition is significantly related to or substantially aggravated by coal dust exposure. *Wilburn v. Director, OWCP,* 11 B.L.R. 1-135 (1988). An equivocal opinion, however, may be given little weight. *Justice v. Island Creek Coal Co.,* 11 B.L.R. 1-91 (1988); *Snorton v. Zeigler Coal Co.,* 9 B.L.R. 1-106 (1986).

The hospitalization records from Mr. Childress's final admission reflect that he was brought to the hospital after suffering a heart attack. He was treated by Dr. Ginsberg, who reported that Mr. Childress was previously treated with antituberculous therapy for a mycobacterium infection, and that when he was admitted, his diagnoses included cavitary lung lesions and diffuse pulmonary infiltrates. During his course at the hospital, Mr. Childress was diagnosed with severe cardiomyopathy, and he underwent angioplasty.

But the only references to pneumoconiosis in these hospital records were Dr. Ginsberg's notations of "presumed pneumoconiosis." Dr. Ginsberg was Mr. Childress's treating physician. He first saw Mr. Childress on referral from Dr. Snell, who felt he should be evaluated for mycobacterial disease. Dr. Ginsberg noted that Mr. Childress had been treated in February 2001 for toxic symptoms of right apical pulmonary abscess, which had been confirmed as mycobacterium avium, and responded to antibiotics. After evaluating Mr. Childress, and reviewing testing, Dr. Ginsberg recommended that Mr. Childress begin therapy for tuberculosis and/or mycobacterium avium complex. He sent Mr. Childress to the Department of Health, where a skin test was positive for tuberculosis.

Dr. Ginsberg's treatment notes contain suggestions of pneumoconiosis, such as his initial comment that bilateral interstitial infiltrates are considered compatible with pneumoconiosis, a notation that pneumoconiosis had been noted on previous x-ray films, and his statement in January 2003 that Mr. Childress presented with pneumoconiosis associated with dust inhalation, as well as mycobacterium simiae. Especially in light of his comments in the hospital records that Mr. Childress had "presumed" pneumoconiosis, I find that Dr. Ginsberg's opinion on this issue is not supported by any rationale or objective evidence, and I accord it little weight.

Dr. Chamberlain saw Mr. Childress on referral from Dr. Ginsberg, and noted that he had been diagnosed with tuberculosis a year earlier. He concluded that Mr. Childress had left vocal cord paralysis and tuberculosis, but he made no findings of pneumoconiosis.

Dr. Snell reviewed Mr. Childress's CT scan of May 2003, concluding that it confirmed a pattern more consistent with a granulomatous infection than pneumoconiosis. He had also requested sputum cultures, which showed that Mr. Childress had a recurrent infection of inadequately treated mycobacterium simae. According to Dr. Snell, it was very clear that Mr. Childress did not have an occupational lung condition, but he had an infection that had flared up for years, and gotten out of control.

In addition to reviewing the autopsy slides, Dr. Caffrey reviewed Mr. Childress's medical records, and stated that there was no question that Mr. Childress developed mycobacterium avium and/or mycobacterium simae. He was not aware of any association between these conditions and pneumoconiosis.

Dr. Mull completed Mr. Childress's death certificate, indicating that pneumoconiosis was a significant condition contributing to his death. But Dr. Mull did not have any autopsy findings available. Nor is there any indication that Dr. Mull ever treated Mr. Childress, or had access to any of his medical records. There is no indication of the basis for his notation of pneumoconiosis. I find that Dr. Mull's notation is not a reliable basis for a finding of pneumoconiosis.

Thus, Dr. Ginsberg is the only physician who has suggested that Mr. Childress had pneumoconiosis. But his notations are unsupported by any objective evidence, or any rationale, save for his statement that bilateral infiltrates are compatible with pneumoconiosis. In fact, Dr. Ginsberg treated Mr. Childress for his mycobacterium tuberculosis. I find that his conclusory notations of pneumoconiosis and "presumed" pneumoconiosis are not a reliable basis for a finding of pneumoconiosis.

Accordingly, I find that the Claimant has not established that Mr. Childress had pneumoconiosis by a preponderance of the reliable medical opinion evidence.

Finally, I have weighed all of the evidence under § 718.202(a), including the x-ray evidence, and I find that the Claimant has not met her burden to establish that Mr. Childress had pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 2000 WL 524798 (4th Cir. 2000). As the Claimant has not established the existence of pneumoconiosis, she is not entitled to benefits under the Act.

Living Miner's Claim4

X-Ray Evidence

In connection with Mr. Childress's living miner's claim, the record includes the following x-rays.

Exhibit No.	***Date of X-Ray***	***Date of Reading***	***Physician/ Qualifications5***	***Interpretation***
DX 13	3-25-00	3-25-00	Ghearing	Interstitial densities throughout both

4 At the hearing, there was much discussion of the status of Mr. Childress's living miner's claim, which affects the amount of evidence the parties are allowed to submit. Although I initially advised the parties that this claim is a modification, I subsequently concluded that it was an initial claim. After closer review of the record, I find that my initial determination was correct: this claim is a modification of the Director's denial of , which became final after Mr. Childress did not request an appeal. The parties advised me at the hearing as to what evidence they wished to be considered on a modification, and I have considered that evidence.

5 A "B-reader" is a physician, but not necessarily a radiologist, who has successfully completed an examination in interpreting x-ray studies conducted by, or on behalf of, the Appalachian Laboratory for Occupational Safety and Health (ALOSH). A designation of "BCR" means that the physician is "certified" in radiology or diagnostic roentgenology by the American Board of Radiology or the American Osteopathic Association.

				lungs, probably related to inhalational lung disease
DX 13	2-2-01	2-2-01	Willis	Diffuse bilateral interstitial disease, with varying size small round opacities, and some densities in periphery of mid lungs
DX 16	3-9-01	3-9-01	Ginsberg	Micronodular and macronodular reticular infiltrate throughout lung zones
DX 16	4-3-01	4-3-01	Ginsberg	Collateral interstitial macronodular infiltrate; air fluid level in right upper lobe gone. Diaphragms elevated, associated with restrictive ventilatory impairment or reduced lack of effort for inspiration; mass effect not appreciated but could be obscured
DX 12	7-20-01	7-20-01	Hudson	2/3, q, r
DX 12	7-20-01	9-11-01	Sargent/B, BCR	Read for quality only
DX 14	7-20-01	11-19-01	Wheeler/B, BCR	0/1, q, t
DX 16	10-9-01	10-9-01	Ginsberg	Diffuse macronodular infiltrate in all areas of the lung; air fluid level in right upper lung zone no longer present; elevated diaphragms suggesting restrictive ventilatory impairment
DX 19	11-15-02	4-11-03	Scatarige/B, BCR	Negative for pneumoconiosis
DX 48	11-15-02	12-13-02	Ahmed/B, BCR	3/3

Pulmonary Function Studies

Exhibit No.	***Date***	***Age/Ht***	***FEV1***	***FVC***	***MVV***	***Effort***	***Qualifying6***
DX 12	7-20-01	54/66”	2.04	2.51			
DX 14	2-16-01	53/67”	2.08 2.12*	2.38 2.46*			
DX 14	3-9-01	53/68”	2.14	2.64			

6 A “qualifying” pulmonary function study yields values that are equal to or less than the appropriate values set out in the tables at 20 C.F.R. Part 718, Appendix B. A “non-qualifying” study exceeds those values. 20 C.F.R. §718.204(b)(2)(i).

Exhibit No.	*Date*	*Age/Ht*	*FEV1*	*FVC*	*MVV*	*Effort*	*Qualifying6*
DX 15	3-20-01	54/68"	2.87 2.91*	3.57 3.45*	84 90*	Good	
DX 16	10-9-01	54/68"	1.95	2.56			
DX 17	1-10-03	55/68"	1.63	2.23			

* Results after administration of bronchodilators

Arterial Blood Gas Studies

Exhibit No.	*Date*	*Physician*	*pCO2*	*pO2*	*At rest* *After exercise*
DX 127	7-20-01	Hudson	35.9 35.7	75 54	At rest After exercise
DX 14	3-20-01	Ginsberg	36	75.1	At rest

Medical Opinion Evidence

The record in Mr. Childress's living miner's claim includes the reports by Dr. Richard L. Naeye, Dr. James D. Snell, the treatment notes from Dr. Joel Ginsberg, and the report by Dr. Morrow Chamberlain, as summarized above. In addition, the record includes the following.

Dr. A.R. Hudson

Dr. Hudson examined Mr. Childress on July 20, 2001 at the request of the Department of Labor (DX 12). He reported Mr. Childress's coal mine employment history, as well as his family and medical histories. Mr. Childress told Dr. Hudson that he smoked about a pack a day of cigarettes from his late teens until 1978.

On examination of Mr. Childress, Dr. Hudson noted scattered rhonchi on auscultation, which cleared with cough, and faint mid pitched bilateral inspiratory wheezes. Mr. Childress informed Dr. Hudson that he had undergone extensive evaluation for an abnormal chest x-ray by Dr. Ginsberg, which sounded to Dr. Hudson as if he had a chest CT and needle biopsy. Mr. Childress's understanding was that his diagnosis was coal workers' pneumoconiosis.

7 Dr. Michos reviewed the results of this study, and found them to be technically acceptable.

Dr. Hudson administered an x-ray, which was positive, and pulmonary function and arterial blood gas testing. He concluded that Mr. Childress had a restrictive ventilatory impairment with diffuse nodular infiltrates, consistent with simple pneumoconiosis. Assuming that previous evaluations were negative for other etiologies, he attributed this condition to Mr. Childress's coal mining. He noted that Mr. Childress provided a history of adequate mining exposure to cause pneumoconiosis, and no history of other causes of interstitial lung disease. Dr. Hudson felt that Mr. Childress's impairment was moderate, and that he was capable of light work.

Dr. Sarah B. Long

Dr. Long reviewed the pulmonary function study performed on January 10, 2003 (EX 3). She noted that the name of the instrument used was not provided; there was a signature by a technician, but there was no physician's name or signature. There were three flow tracings, but no flow volume loops. The tracings showed significant differences in the curves. There were three spirometric tracings, but they were recorded at 10 mm/sec. instead of the 20 mm/sec. required by the regulations. She noted that the curves were superimposed, so that evaluation of the individual tracings was not possible. She noted significant variation in the best two FEV1 values, indicating inconsistent effort. However, the values could not be calculated from the superimposed curves.

According to Dr. Long, the study is not valid because of these deficiencies, and would not be useful in the evaluation of a respiratory impairment.

Treatment Records

Erlanger Health System

Mr. Childress was admitted to the Erlanger Health System on February 15, 2001, and discharged on February 21, 2001 (DX 16). He had been transferred from Dr. McCartney's clinic after failing outpatient treatment for pneumonia. A recent x-ray was of concern for a bilateral interstitial process. In the past medical history, Dr. Herman noted a question of black lung, and hypertension of twenty years. Mr. Childress was admitted and treated with antibiotics for multi-lobar pneumonia on top of an interstitial lung disease process, and abscess versus cavitary neoplasm in the right upper lobe. Dr. Herman was not sure if Mr. Childress had an atypical pneumonia, or possibly a fungal process.

Dr. Ginsberg saw Mr. Childress on consultation, and recommended a high resolution CT scan, which showed extensive air space disease in the right upper lobe with central air fluid level that was contiguous with the mediastinum, and extended to the pleural surface and into the superior right lower lobe. There were also left lobe pleural based opacities. The differential diagnosis was necrotic neoplasm, versus infection, versus tuberculosis or fungal infection.

Mr. Childress underwent a CT guided aspiration and core biopsy. There was no evidence of malignancy, pneumocystitis or AFB, but the core biopsy was not satisfactory for diagnosis.

Mr. Childress's pulmonary function studies showed restrictive lung disease, FEV1 of 2, FVC of 2.38. Mr. Childress's condition improved, and after his CT scan was interpreted as possible tuberculosis, he was placed in isolation until after he had three negative AFBs. His x-ray continued to show diffuse bilateral reticular nodular interstitial infiltrates, possibly due to interstitial lung disease versus TB or a fungal process. The cavitary lesion that had been seen in the right upper lobe was no longer seen, nor was the air fluid level. He was discharged, and advised to remain off work until the fungal and TB cultures had grown, in case he had a mycobacterial infection.

Dr. Joel Ginsberg

Mr. Childress made his first visit to Dr. Ginsberg on March 9, 2001. Dr. Ginsberg reported that Mr. Childress had a 25 pack year history of smoking, and had experienced a subtle change in his pulmonary status over the previous two years.8 A chest x-ray had shown bilateral interstitial infiltrate of uncertain etiology, perhaps related to exposure to coal dust.

Dr. Ginsberg reported that on admission to Erlanger in February 2001, Mr. Childress's x-ray showed an airfluid level, and complex pleural and parenchymal thickening in the right apex. There was extensive air space disease, macro and micronodular. The CT scan showed an extensive interstitial process throughout both lung fields, and an acute appearing consolidation surrounding a thin walled cystic structure with air fluid level, contiguous with the mediastinum, and extending to the pleural surface and into the superior right lower lobe. There were also left lower lobe pleural based opacities. The differential diagnosis was necrotic neoplasm versus infection versus tuberculosis or fungal etiologies. A CT guided aspiration showed no malignancy, pneumocystitis, or AFB. Dr. Ginsberg indicated that there was eventually a report of mycobacterium avium.

On this date, Mr. Childress was much improved. His chest x-ray showed no air fluid level in the right apex, although there were micronodular and macronodular infiltrates in the periphery of the lung fields. His pulmonary function studies showed restriction, and a small component of obstructive ventilatory impairment. Dr. Ginsberg continued Mr. Childress on antibiotics, and awaited AFB cultures; antituberculous therapies were under consideration.

Mr. Childress underwent a CT scan of his thorax on March 9, 2001. Dr. Thomas, who reviewed the scan, concluded that it showed bilateral upper lobe bronchiectasis, as well as an extensive reticulonodular pattern throughout both lungs, with volume loss in the upper lobes, consistent with his given diagnosis of tuberculosis. She indicated that on a previous CT scan, there was a cavitary lesion in the right upper lobe, but there was no fluid level on this study. She noted a necrotic paratracheal adenopathy, which had been present on the previous study but obscured by the large cavitary mass. Dr. Thomas also reviewed a chest x-ray of the same date, noting extensive bilateral reticular nodular changes consistent with the given diagnosis of tuberculosis.

Dr. John Galbraith reviewed a CT scan of Mr. Childress's thorax that was performed on March 16, 2001 at Dr. Ginsberg's request. He noted a decrease in the opacification in the right

8 Dr. Ginsberg stated in the same report that Mr. Childress smoked for 50 years.

upper lobe, as well as resolution of a cavitary mass. There was still extensive bilateral pulmonary disease in the upper portions of both lungs, with some improvement in the right upper lobe. A necrotic node was also somewhat smaller. He noted a very minimal degree of scattered pleural thickening. Dr. Galbraith felt that the disease pattern was quite suggestive of a granulomatous disease, possibly active TB.

Dr. Ginsberg also requested pulmonary function studies, which were performed on March 20, 2001. According to the report, the lung mechanics showed no evidence of significant obstructive airways disease. There was no change in the lung volumes after administration of bronchodilators. Dr. Ginsberg felt that the lung volumes were most consistent with mild chest restriction, although he questioned the reliability of the studies, as the slow vital capacity was over a half a liter than FVC. The diffusion capacity was moderately to severely reduced, which was also a doubtful result, given the low measured alveolar ventilation. Finally, the normal resting blood gases were curiously elevated after administration of bronchodilators.

Dr. Ginsberg saw Mr. Childress on April 3, 2001. He noted that a call to the Erlanger Microbacteriology Laboratory on March 20 had indicated an unidentified acid fast bacillus, either mycobacterium tuberculosis or mycobacterium avium. Mr. Childress's chest x-ray showed micronodular and micronodular infiltrates in the periphery of the lung zones. His pulmonary function tests showed the absence of obstructive or restrictive ventilatory impairment, although the lung volumes showed a mild restrictive impairment associated with a significant reduction in carbon monoxide transfer, most likely representing pneumoconiosis. The arterial blood gas results showed minimal hypoxemia.

According to Dr. Ginsberg, Mr. Childress did not have signs or symptoms of air fluid formation in his right apex. There was a complex pneumoconiosis appearance on the x-ray. Dr. Ginsberg did not prescribe additional antibiotic therapy, pending the results of final growth identification of the mycobacterium organism.

Dr. Ginsberg saw Mr. Childress on October 9, 2001. His pulmonary function studies showed combined moderate obstructive and restrictive ventilatory impairment, associated with coal workers' pneumoconiosis. Dr. Ginsberg reported that the right apical changes had resolved, and evidence of coal miners' pneumoconiosis had again been identified.

Discussion

Existence of Pneumoconiosis

In the living miner's claim, there are four ILO interpretations of two x-rays. The first, performed on July 20, 2001, was interpreted as positive by Dr. Hudson, who is neither a B reader nor board certified radiologist, and as negative by Dr. Wheeler, who is dually qualified. Dr. Wheeler also interpreted the November 15, 2002 x-ray as negative, but Dr. Ahmed, who is dually qualified, interpreted it as positive. Given the preponderance of negative readings by the most highly qualified physicians, I find that the Claimant has not established that Mr. Childress had pneumoconiosis by a preponderance of the x-ray evidence.

Neither the Claimant nor the Employer designated any of the autopsy evidence in connection with the living miner's claim. Had they done so, however, I would make the same findings as in the survivor's claim, that is, that Dr. Emerson's limited findings are not sufficient to support a finding of pneumoconiosis.

I have discussed the reports by Dr. Naeye, Dr. Ginsberg, and Dr. Chamberlain in connection with the survivor's claim.9 Dr. Hudson examined Mr. Childress in connection with his living miner's claim. He concluded that Mr. Childress had a restrictive ventilatory impairment consistent with simple pneumoconiosis. However, for a number of reasons, I find that Dr. Hudson's conclusions are not reliable.

Dr. Hudson reported that Mr. Childress told him he had undergone extensive evaluation for an abnormal chest x-ray by Dr. Ginsberg, and his understanding was that he was diagnosed with coal workers' pneumoconiosis. In fact, Dr. Ginsberg had diagnosed and treated Mr. Childress for a form of tuberculosis. There is nothing in Dr. Hudson's report to indicate that he was aware of Mr. Childress's extensive history with this infectious condition. Indeed, Dr. Hudson conditioned his finding that Mr. Childress's respiratory condition was due to his coal mining employment on his assumption that previous evaluations were negative for other etiologies, when demonstrably, they were not. Dr. Hudson explicitly relied on the fact that Mr. Childress had an adequate history of mining exposure to cause pneumoconiosis, and no history of other causes of interstitial lung disease. But Mr. Childress did have a significant history of a rare form of tuberculosis, which resulted in significant interstitial lung disease.

Thus, Dr. Hudson's conclusions were based on the assumption that there was no other potential cause for the abnormalities on Mr. Childress's x-ray, or for his restrictive impairment. As his assumption was incorrect, I find that his conclusions are not reliable evidence of pneumoconiosis.

The records from Mr. Childress's admission to the Erlanger Health System do not persuasively support a conclusion that he had pneumoconiosis. He was admitted for treatment of pneumonia, and CT scans, x-rays, and core biopsy resulted in differential diagnoses of neoplasm, interstitial lung disease, or tuberculosis or fungal infection. The only mention of pneumoconiosis was in Mr. Childress's medical history, where the admitting physician indicated that there was a "question" of black lung.

After he was discharged from Erlanger, Mr. Childress began treatment with Dr. Ginsberg. Dr. Ginsberg's records reflect that eventually, it was determined that Mr. Childress had a form of tuberculosis called mycobacterium avium, and he treated him with antibiotics. CT scans ordered by Dr. Ginsberg produced findings consistent with tuberculosis. In his April 3, 2001 treatment note, Dr. Ginsberg stated that Mr. Childress's pulmonary function tests produced lung volumes that showed a mild restrictive impairment associated with a significant reduction in carbon monoxide transfer, which most likely represented pneumoconiosis. He also noted a "complex pneumoconiosis appearance" on x-ray. In his note of October 9, 2001, Dr. Ginsberg stated that the obstructive and restrictive impairment on pulmonary function studies was associated with

9 I have not considered the report by Dr. Caffrey, as it would exceed the Employer's evidentiary limitations.

coal workers' pneumoconiosis, and that evidence of coal miners' pneumoconiosis had again been identified.

I find that Dr. Ginsberg's statements are too vague and speculative to support a finding that Mr. Childress had pneumoconiosis, especially in light of the fact that the preponderance of the x-ray evidence is negative for pneumoconiosis, and the persuasive autopsy evidence does not establish pneumoconiosis. Dr. Ginsberg did not explain why the findings on pulmonary function studies supported a diagnosis of pneumoconiosis. It is not at all clear what he was referring to when he stated that evidence of coal miners' pneumoconiosis had been identified, or whether this referred to Dr. Hudson's x-ray interpretation several months earlier.

Given the overwhelming medical evidence establishing that Mr. Childress suffered from a rare form of tuberculosis, and the corresponding dearth of persuasive evidence to support a finding of pneumoconiosis, I find that the Claimant has not established that Mr. Childress had pneumoconiosis by a preponderance of the medical opinion evidence.

Again reviewing all of the evidence relating to the existence of pneumoconiosis as a whole, I find that the Claimant has not established that Mr. Childress had pneumoconiosis.

CONCLUSION

The Claimant has not established that the miner, Clyde Childress, suffered from pneumoconiosis that arose out of his coal mine employment. Therefore, the Claimant is not entitled to benefits under the Act on either the living miner's or the survivor's claim.

ORDER

It is hereby ORDERED that the survivor's claim for benefits under the Act is hereby DENIED, and that the miner's claim for benefits under the Act is hereby DENIED.

SO ORDERED.

A

LINDA S. CHAPMAN
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of

Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).